U.S. Department of Labor

Office of Administrative Law Judges
Seven Parkway Center - Room 290
Pittsburgh, PA 15220

(412) 644-5754
(412) 644-5005 (FAX)

Issue Date: 12 July 2006

CASE NO. 2002-BLA-389

In the Matter of

RUTH L. BOWERSOX, Survivor of
KENNETH BOWERSOX,
Claimant

v.

MARK MINING II, INC.,
Employer

and

OLD REPUBLIC INSURANCE CO.,
Carrier

and

DIRECTOR, OFFICE OF WORKERS'
COMPENSATION PROGRAMS
Party-in-Interest

DECISION AND ORDER ON REMAND ─ AWARDING BENEFITS

This proceeding arises from a claim for benefits under the Black Lung Benefits Act, 30 U.S.C. §§ 901–945 ("the Act") and the regulations issued thereunder, which are found in Title 20 of the Code of Federal Regulations. Benefits under the Act are awarded to coal miners who are totally disabled within the meaning of the Act due to pneumoconiosis, or to the survivors of coal miners whose death was due to pneumoconiosis. Pneumoconiosis, commonly known as black lung, is a dust disease of the lungs resulting from coal dust inhalation.

Following a formal hearing held before Administrative Law Judge Robert J. Lesnick on December 9, 2002, in State College, Pennsylvania, Judge Lesnick issued a Decision and Order - Awarding Benefits on September 8, 2003. The employer appealed to the Benefits Review Board ("the Board"), which issued a Decision and Order on November 14, 2004, affirming Judge Lesnick's finding that the evidence establishes that the miner's death was due to pneumoconiosis pursuant to 20 C.F.R. § 718.205(c).

Employer then filed a Motion for Reconsideration with Suggestion of Reconsideration En Banc of the Board's Decision and Order in *Bowersox v. Mark Mining II, Inc*., BRB No. 04-0103

BLA (November 18, 2004)(unpub.). The Board issued its Decision and Order on Motion for Reconsideration on March 14, 2005, which vacated its affirmance of Judge Lesnick's Decision and Order Awarding Benefits. As Judge Lesnick is no longer with the Office of Administrative Law Judges, the case was assigned to me.

On reconsideration, the Board found that Judge Lesnick did not specifically address whether the opinions of Drs. Perper and Rizkalla are sufficiently reasoned to constitute substantial reliable and probative evidence that the miner's pneumoconiosis hastened his death. Consequently, the Board vacated its previous Decision and Order only insofar as it affirmed Judge Lesnick's finding that the opinions of Drs. Perper and Rizkalla are reasoned, and the Board also vacated Judge Lesnick's finding that Claimant established that the miner's death was due to pneumoconiosis pursuant to 20 C.F.R. § 718.205(c).

The Board remanded the case to Judge Lesnick for further consideration consistent with its opinion and instructed him to consider the opinions of Drs. Rizkalla and Perper and to "specifically explain whether each physician has provided a reasoned opinion." BRB Decision and Order on Reconsideration, p. 2.

The summary of medical evidence in Judge Lesnick's Decision and Order - Awarding Benefits, issued September 8, 2003, is incorporated by reference; however, pertinent facts will be restated for clarity of discussion and in conformance with the Board's instruction in its Decision and Order.

Medical Reports

Dr. Perper

Dr. Joshua A. Perper, a pathologist, reviewed the miner's medical records, the medical consulting opinions, and the autopsy report, slides, and death certificate. His report appears in the record at both DX 23 and 39. Dr. Perper's microscopic diagnoses are: 1) Simple coal workers' pneumoconiosis; 2) Centrilobular emphysema and interstitial fibrosis, more peripheral, moderate to severe; 3) Acute bronchopneumonia, lower lobes; 4) Sclerosis of small intra-pulmonary blood vessels. Dr. Perper opined that the miner had an occupational lung disease as a result of his coal mine exposure. He based this diagnosis on the miner's clinical symptomatology consistent with COPD that required the use of respiratory inhalers and oxygen, and emphasized that the miner's ventilatory studies showed a restrictive component and hypoxia, in addition to an obstructive element, which he deemed "a combined obstructive and restrictive lung disease." Dr. Perper explained that such restrictive impairment is not seen with emphysema that shows only an obstructive component but it is seen in coal workers' pneumoconiosis that shows both an obstructive and restrictive functional impairment.

Dr. Perper acknowledged that the miner was a former smoker but explained that as abundantly substantiated in reliable scientific literature in recent decades, centrilobular emphysema is also a direct result of exposure to mixed coal mine containing silica and coal workers' pneumoconiosis. Dr. Perper provided an appendix with citations to several textbooks and medical journals, both British and American, in support of this statement. Dr. Perper stated

that it is logical to recognize the role of coal dust exposure and coal workers' pneumoconiosis in producing centrilobular emphysema, as much as smoking, and that there is no logical reason to exclude it. Dr. Perper explained that the scientific literature recognizes exposure to coal mine dust and centrilobular emphysema as being more significant than any effect that may be attributed to smoking, and that coal workers' pneumoconiosis and its emphysema complications progress after ending exposure to coal dust, because of the entrapped and retained intrapulmonary fibrogenic crystalline silica.

Dr. Perper noted that although the chest x-ray readings did not indicate simple coal workers' pneumoconiosis, some of them noted an opacity described as large as 1-1.5 cm and believed to be a granuloma. He noted that no granulomas were found on the miner's autopsy and explained that in his experience of the majority of cases he autopsied or reviewed, if there is no clinical documentation of tuberculosis or other granulomatous disease, the so-called "granulomas" prove to be pneumoconiotic lesions on biopsy or autopsy.

Dr. Perper opined that the miner had evidence of moderate to severe centrilobular emphysema and interstitial fibrosis. He noted that the miner was both a past smoker and a coal miner exposed to silica containing coal dust and both exposures are associated with the development of centrilobular emphysema and related COPD. Dr. Perper explained that the causal connection between exposure to coal and silica with respect to emphysema and chronic obstructive lung disease is widely and universally accepted and cited the textbook, "Diseases of the Chest", second edition and an article in the New England Journal of Medicine in support of this statement. Dr. Perper also explained that recent literature substantiates that the pulmonary damage associated with exposure to coal mine dust and CWP may progress even after cessation of occupational exposure to silica containing mixed coal mine dust and noted that inhaled silica remains trapped in the lungs and cannot be removed. He cited several other medical journals and texts in support of this statement.

Dr. Perper opined that the miner's coal workers' pneumoconiosis and associated centrilobular emphysema contributed to his death and hastened his demise. He based this opinion on: 1) Clinical respiratory manifestations of shortness of breath, cough, and expectoration of mucus; 2) Manifestations of respiratory problems before any manifestations of peripheral edema and no evidence at autopsy of myocardial ischemic changes or myocardial infarction; 3) Clinical diagnosis of chronic obstructive pulmonary disease, requiring bronchodilators and oxygen; 4) Laboratory test indicative of obstructive defect, restrictive defect, and hypoxemia; 5) Pathological findings at autopsy indicative of significant simple coal workers' pneumoconiosis and associated centrilobular emphysema and interstitial fibrosis; 6) Presence at autopsy of sclerosis of small intra-pulmonary blood vessels consistent with pulmonary hypertension and cor pulmonale; 7) The autopsy finding of an 0.8 cm thickness of right ventricular wall, a finding indicative of hypertrophy of right ventricular mass and cor pulmonale; 8) The miner suffered severe shortness of breath, was steroids dependent and ultimately oxygen dependent.

Dr. Perper stated that the immediate cause of death was acute bronchopneumonia complicating the miner's chronic lung disease, due in significant part to his exposure to coal mine dust and resulting pneumoconiosis. He opined within a reasonable degree of medical

certainty that: 1) The miner acquired an occupational disease, coal workers' pneumoconiosis, as a result of his long standing exposure as an underground coal miner; 2) The degree of the miner's coal workers' pneumoconiosis was substantial and was associated with centrilobular emphysema and chronic obstructive pulmonary disease; 3) The miner's coal workers' pneumoconiosis was a substantial factor in causing, substantially contributing to, or hastening his death.

Dr. Perper testified in a deposition on October 7, 2002. The deposition transcript appears in the record at CX 2. Dr. Perper defined and described aspiration pneumonia and testified that there was no significant or insignificant aspiration pneumonia in the miner's slide material or any kind of pneumonia in which he could identify the fragments that caused the process, whether acute or chronic. CX 2 at pp. 16-18. He explained that Dr. Oesterling was the only physician that diagnosed aspiration pneumonia. CX 2 at p. 19. He testified that he looked at the same photograph, which showed a food particle less than one sixteenth of an inch, and that there was no inflammation around it, a particle of that size is absolutely and totally insignificant, and the presence of a particle does not constitute aspiration pneumonia. CX 2 at pp. 20-21. Dr. Perper testified that he observed pneumonia in nine different slides but there were no aspirated food particles as the cause of the pneumonia. CX 2 at pp. 22-23. He explained that when aspiration is the cause of a fatal pneumonia it is obvious at autopsy, one will see the presence of the aspirated gastric material, and there are going to be chronic inflammatory cells in the presence of multi-nuclear giant cells. CX 2 at p. 42.

Dr. Perper described the processes he found in the lung tissue, which included fibroanthracosis, birefringent crystals, tiny micronodules, anthracotic and silica pigmentation, fibroanthracotic micro nodules, focal emphysema, centrilobular emphysema, and interstitial fibrosis, that he deemed were consistent with occupational pneumoconiosis. CX 2 at pp. 23-24. He testified that there was also thickening and sclerosis of the small blood vessels inside the lungs, which is an indication consistent with pulmonary hypertension and cor pulmonale, along with areas of acute bronchopneumonia, edema, and focal hemorrhages that were present in the lower lobes. CX 2 at p. 24. Dr. Perper explained that the coal workers' pneumoconiosis and associated centrilobular emphysema are the type of lung disease that predisposes to pneumonia. CX 2 at p. 25. He stated that the miner had sclerotic changes of the blood vessel consistent with pulmonary hypertension and cor pulmonale, and that there were also areas that showed silicotic nodules in the lymph nodes of the lungs with numerous birefringent crystals. *Id.* He noted that these did not appear in the micrographs of Dr. Oesterling and they are indicative of substantial severe exposure to a dust atmosphere that contains coal and silica. *Id.*

Dr. Perper opined that the cause of the extensive acute bronchopneumonia was the propensity of the miner to develop infectious disease because of his chronic lung disease that was primarily due to coal workers' pneumoconiosis and coal dust exposure. CX 2 at p. 27. Dr. Perper testified that the miner's cigarette smoking contributed to his moderately severe centrilobular emphysema and his coal dust exposure also contributed. CX 2 at p. 33. He testified that there is no way to exclude either cigarette smoking or coal dust exposure from the emphysematous burden at the time of the miner's death but explained that once a person stops smoking there is no further deterioration of respiratory function, except for deterioration that is age related and those that are very slowly progressing. *Id.* Dr. Perper stated that the miner's

case demonstrated a much more significant progression of respiratory symptoms after he stopped smoking, which is consistent with coal workers' pneumoconiosis because it progresses after cessation of exposure to coal dust. CX 2 at pp. 33-34. Dr. Perper testified that the coal workers' pneumoconiosis was of sufficient severity by the detailed pathological finding and the gross and microscopic pathological findings described by the prosector to account for the accompanying significant centrilobular emphysema. CX 2 at p. 36.

Dr. Perper testified that there was concern in the clinical record about the miner's susceptibility to aspirating food, which was due to his two neurological disorders, Parkinson's and Alzheimer's disease. CX 2 at p. 39. He explained that the miner's clinicians tested him for it and there was no evidence of aspiration pneumonia at the autopsy, so the clinical treatment and worry about it most likely prevented it from happening. *Id.* Dr. Perper testified that there is no objective way to exclude the miner's pre-existing underlying centrilobular emphysema and coal workers' pneumoconiosis from substantially contributing to his respiratory death. CX 2 at p. 40.

Dr. Perper testified that although renal failure was listed as the cause of death on the death certificate, in his opinion it was not ultimately determined by any of the pathologists to be the cause of death. CX 2 at p. 43. He explained that there was an increased manifestation of renal dysfunction terminally in an individual with pulmonary disease - the acute bronchopneumonia in the background of coal workers' pneumoconiosis and emphysema. *Id.* Dr. Perper explained that Parkinson's Disease was not a cause of death and that many times clinicians list a number of diagnoses that they think are significant but are not necessarily a cause of death. CX 2 at p. 44. He stated that aspiration pneumonia, which was also listed as a contributory cause of death could be a cause of death, but in this particular case there was bronchopneumonia but no aspiration pneumonia. *Id.* Dr. Perper testified that COPD was correctly identified on the death certificate because the proper certification of death should have said, "acute bronchopneumonia due to chronic obstructive pulmonary disease, due to coal workers' pneumoconiosis and centrilobular emphysema." CX 2 at p. 45.

Dr. Perper testified that the supplemental medical literature he provided with his report is to indicate that the processes he identifies in his report are not only a result of his experience but also are recognized processes in the medical literature. CX 2 at p. 46. Dr. Perper stated that although the vegetable material was located in the right lower lobe and the area where the prosector diagnosed pneumonia was also in the right lower lobe, it doesn't make it an aspiration pneumonia. CX 2 at p. 51. He explained that it is similar to a building with many stores, and if in one room there is a piece of destroyed furniture, it does not mean that there is destroyed furniture in all of the other rooms of the building. *Id.* He explained that one cannot make a diagnosis of aspiration pneumonia without seeing the aspiration in the midst of the acute inflammatory infiltrates. CX 2 at p. 53. Dr. Perper testified that he diagnosed simple coal workers' pneumoconiosis and that there was no progressive massive fibrosis present. CX 2 at p. 56. He testified that no other reviewing pathologist reviewing the case found evidence of confluence nodules. CX 2 at p. 58. Dr. Perper explained that anthracotic pigmentation can occur in urban dwellers who are not exposed to coal dust but those individuals do not have silica in the area of the anthracotic pigmentation. CX 2 at p. 60. Dr. Perper testified that he did not identify the lesions in the lower lung lobes and explained that is something that's precisely the area in which characteristically lesions of coal workers' pneumoconiosis are present. CX 2 at p. 63.

Dr. Perper testified that in this particular case, the findings are consistent with cor pulmonale and explained that the prosector described a thickness of the right ventricle of 0.8 cm, and this was confirmed with the sclerotic changes he saw in the lungs and are consistent with pulmonary hypertension. CX 2 at p. 66. He testified that he did not make this diagnosis in his report because he didn't measure the right ventricular wall and stated that the autopsy prosector did not diagnose cor pulmonale. *Id.* Dr. Perper testified that the miner's left ventricle was also enlarged and explained that when there is right ventricular hypertrophy secondary to left ventricular hypertrophy, you don't get the pulmonary hypertension and changes of pulmonary hypertension in the lung that you get with chronic obstructive lung disease or any pulmonary process. CX 2 at p. 69.

Dr. Perper testified that he considered that the miner had a significant smoking history of between ten and thirty years and that thirty years was significant enough that he did not need to consider more years than that. CX 2 at pp. 74-75. Dr. Perper stated that in this particular case, the miner had a concomitant exposure to smoking and coal dust. CX 2 at p. 76. He explained that after the cessation of smoking, you can't differentiate between the two exposures and then following the cessation of smoking there is an increased progression that would not have occurred with smoking alone. *Id.* Dr. Perper testified that pneumoconiosis is a progressive disease and that theoretically, if a restrictive defect were present, one would expect to see the defect in later tests. CX 2 at p. 85. He explained that in practice he has seen cases of coal workers' pneumoconiosis where the initial restrictive changes were not sustained or observed and his explanation is that perhaps the obstructive element totally overshadows the restrictive element. *Id.*

Dr. Perper testified that there is no typical thing for coal workers' pneumoconiosis in symptomatology and one cannot make a diagnosis of coal workers' pneumoconiosis based on the symptoms but must add to the symptoms the other elements which include laboratory findings and especially pathological findings. CX 2 at pp. 92-93. He explained that there are symptoms that are often seen with coal workers' pneumoconiosis and they are consistent with coal workers' pneumoconiosis when they are congruent with other findings. CX 2 at p. 93. Dr. Perper testified that the emphysema was greater in severity than the coal workers' pneumoconiosis and explained that one doesn't need a severe condition to get a severe complication but can have a moderate condition and get a severe condition, and sometimes even a slight condition can result in a severe complication. CX 2 at p.95.

Dr. Rizkalla

Dr. Waheeb Rizkalla, who is also a pathologist, reviewed the miner's medical records, including the other medical consulting opinions, the autopsy report, death certificate, and the microscopic slides from the miner's autopsy. He issued a consultation report dated March 22, 2002. The report appears in the record at DX 41. Dr. Rizkalla recorded the miner's clinical history as primarily deep coal mining and coal mine work for approximately forty years. He noted that the miner smoked for approximately twenty years and quit prior to his death. Dr. Rizkalla noted that the miner's medical history included respiratory ailments with the diagnoses of moderate ventilatory deficit and chronic pulmonary disease, that multiple chest x-rays displayed interstitial infiltrates, and that the miner underwent multiple clinical evaluations

for pulmonary disease, while also receiving numerous treatments with bronchodilators and other methods to relieve his symptoms. Dr. Rizkalla recorded other significant medical issues as transurethral resection of the prostate, and a diagnosis of dementia that was most likely secondary to Alzheimer's disease.

Dr. Rizkalla noted that the autopsy revealed simple coal workers' pneumoconiosis in the form of anthrasilicotic deposits in the lungs in the perivascular and peribronchial areas and the interstitium. He observed that these deposits formed micronodules and macronodules as described by the original prosector, and that the anthrasilicotic pigment contained birefringent needle-shaped crystals consistent with silica. Dr. Rizkalla's microscopic description noted acute bronchopneumonia, no foreign body material or aspiration material present in the bronchi or alveolar spaces. He observed that anthrasilicotic pigmentation deposit is seen around the bronchioles, blood vessels, and interstitium with birefringent needle-shaped crystals consistent with silica. Dr. Rizkalla observed hemosiderin laden macrophages and anthrasilicotic pigmentation present in the macrophages and alveolar spaces, and noted that there is intra-alveolar hemorrhage and centrilobular emphysema. He noted that some emphysematous spaces are coalescent and form subpleural blebs. Dr. Rizkalla remarked that the hilar nodes display concentric areas of fibrosis and anthrasilicotic pigmentation and needle-shaped crystals of silica. He noted that the focal micronodular anthrasilicotic deposits range from 3.0 mm to 5.0 mm and the largest nodule seen by the prosector measures up to 1.0 cm.

Dr. Rizkalla's final anatomic diagnoses were: 1) Simple coal workers' pneumoconiosis; 2) Acute bronchopneumonia; 3) Centrilobular emphysema; 4) Atherosclerotic coronary heart disease (prosector's finding); 5) Cor pulmonale (right ventricle 0.8 cm)(prosector's finding). Dr. Rizkalla's clinicopathological summary was that the miner had simple coal workers' pneumoconiosis with centrilobular emphysema and the immediate cause of death is terminal bronchopneumonia complicating the coal workers' pneumoconiosis. He stated that coal workers' pneumoconiosis is considered a substantial contributing factor in the miner's death.

Dr. Rizkalla opined that the miner had coal workers' pneumoconiosis of a moderate degree with centrilobular emphysema. He explained that considering the miner's more than forty year occupational history, the clinical evaluations, and the autopsy information, there is unequivocal evidence that the miner developed coal workers' pneumoconiosis during his life as a result of his exposure to coal dust. Dr. Rizkalla noted that the miner suffered numerous respiratory symptoms and was evaluated and found to have interstitial fibrosis of his lungs as well as chronic obstructive pulmonary disease.

Dr. Rizkalla opined that coal workers' pneumoconiosis was a substantial contributing factor in the miner's death. He explained that the coal worker's pneumoconiosis induced centrilobular emphysema and cor pulmonale, and that the miner developed terminal bronchopneumonia in addition to his emphysema. Dr. Rizkalla stated that no aspiration material in the form of foreign body or food particles was found in the bronchi or pulmonary tissue and the thickened right ventricle (0.8 cm as measured by the original prosector) would have altered the function of the heart and impaired its normal hemodynamics. Dr. Rizkalla noted that the miner had a history of smoking for a number of years and that smoking is known to induce centrilobular emphysema but study groups show that centrilobular emphysema occurs in coal

workers' pneumoconiosis in non-smokers as well as smokers. Dr. Rizkalla opined that the miner developed terminal bronchopneumonia that complicated his emphysematous lungs, with the emphysema being part of his coal workers' pneumoconiosis. He stated that it is his opinion, within a reasonable degree of medical certainty, that coal workers' pneumoconiosis was a substantial contributing factor in the miner's demise.

Dr. Rizkalla testified in a deposition on August 28, 2002. The deposition transcript appears in the record at CX 1. Dr. Rizkalla testified that the miner had coal workers' pneumoconiosis, anthrasilicotic pigment deposit, silica crystals, areas of fibrosis, areas of bronchopneumonia, centrilobular and focal emphysema, and some emphysematous bullae under the subpleural spaces. CX 1 at p.13. He stated that the miner's coal workers' pneumoconiosis was moderately severe. *Id.* He explained that this means that of the cut section of the lung, approximately 50 percent of the lungs will be affected by coal workers' pneumoconiosis. CX 1 at p. 16.

Dr. Rizkalla testified that aspiration pneumonia can occur in two forms, either acute or chronic. CX 1 at p. 18. He explained that if it is chronic, the body reacts with foreign body giant cells, acute inflammation and one will see the foreign material in the lungs and the body reacts by the foreign body forming granulomas and fibrosis. *Id.* He further explained that in the acute form, one would see the foreign body and the body would react with acute inflammation. *Id.* Dr. Rizkalla testified that he did not see any of that in the miner's case. *Id.* Dr. Rizkalla stated that he identified centrilobular emphysema and focal dust emphysema and it was moderately severe. CX 1 at pp. 18-19. He testified that he did not see pulmonary infarction in this case or pulmonary emboli. CX 1 at pp. 19-20.

Dr. Rizkalla testified that he did not identify any aspirated food material in the miner's lungs. CX 1 at p. 25. He testified that the miner was afflicted in the terminal stages of pneumonia. CX 1 at p. 27. Dr. Rizkalla testified that he identified bronchopneumonia in the miner and that the miner had neurological diseases, pulmonary diseases, heart disease, and his lungs were not healthy to begin with. CX 1 at p.27. He explained that since he had emphysema, he has dilatation of the alveolar sacs and the lungs will lose their normal function, which is to expel secretions, and they will be a fertile media for bacteria and organisms to grow. *Id.* Dr. Rizkalla explained that the miner's emphysematous process made his lungs more susceptible to an infection or inflammation. CX 1 at p. 28.

Dr. Rizkalla testified that the causes of the miner's emphysema were his smoking history and his coal dust exposure. CX 1 at pp. 28-29. He testified that he cannot exclude either cause from consideration and that the miner's nodular disease contributed to the emphysema. CX 1 at p. 29. Dr. Rizkalla testified that even assuming the miner had aspiration pneumonia, it would not have detracted from the underlying role of the moderately severe centrilobular emphysema. CX 1 at p. 31. Dr. Rizkalla testified that the autopsy prosector measured the right ventricle as 0.8 centimeters or eight millimeter in thickness, which is twice the maximum thickness of the left ventricle and that indicates a cor pulmonale. CX 1 at p. 35. He testified that the miner's left ventricle exceeded normal limits and he had bi-ventricular hypertrophy. CX 1 at p. 36.

Dr. Rizkalla explained that it is normal to give swallowing tests and take precautions in patients with neurological deficits, like the miner. CX 1 at pp. 38-39. He testified that he did not see any aspiration material. CX 1 at p. 39. Dr. Rizkalla testified that renal failure was not a cause of the miner's death, and that he may have died with it, but not from it. He testified that the miner died a respiratory death. CX 1 at p. 41. Dr. Rizkalla testified that he relied on a 40-year coal mine employment history for the miner. CX 1 at p. 43. He testified that he relied on a 20-year smoking history but was aware that Dr. Tuteur identified a 30-year smoking history. CX 1 at p. 44. He explained that when you reach that level of smoking - 20 to 30 years - is a long history. *Id.* He testified that this would not be enough smoking, by itself, to cause the emphysema in the miner. *Id.* Dr. Rizkalla further testified that a 72-pack year history would be enough history to induce emphysema by itself in another person but it will not be the only etiological factor in that to induce emphysema. CX 1 at p. 45-47.

Dr. Rizkalla testified that if coal workers' pneumoconiosis were causing a restrictive defect, one would expect to see it on each test, but that if later testing did not reveal the restrictive defect it would not mean that the miner does not have coal workers' pneumoconiosis. CX 1 at pp. 48-49. Dr. Rizkalla testified that when he approaches an autopsy slide or an organ, he approaches it with a clear mind and that he doesn't know what he will be looking for. CX 1 at p. 53. He explained that if he saw aspiration material he would have mentioned it, and if he was asked specifically for it, he will mention it if it's not there. *Id.*

After reviewing some photomicrographs, Dr. Rizkalla testified that one finding would be consistent with a single foreign body material in one field on one slide of the lung. CX 1 at p. 72. He explained that it is unlikely to be food from the miner's clear liquid diet because aspirated food will not be only one single foreign body but should be a huge amount of aspirated material and could be pollen from the air. CX 1 at p.73. He testified that it would not be enough to specify his bronchopneumonia as aspiration pneumonia. CX 1 at p. 91.

Dr. Rizkalla testified that coal dust exposure might predispose one to the bronchopneumonia that he identified, but would not cause it. CX 1 at pp. 74-75. Dr. Rizkalla testified that if he had seen an area of confluent nodulation, he would have mentioned it in his report. CX 1 at p. 79. Dr. Rizkalla explained that after cessation of exposure to coal mine dust, there will be no more coal dust deposited in the lungs but the effect of the coal dust and the silica will keep inducing its effect. CX 1 at p. 82. Dr. Rizkalla testified that the primary lung disease that led to the miner's death was pneumonia. CX 1 at p.83. He explained that if he just had pneumonia and was a normal person with healthy lungs, his chances of dying from pneumonia are very slim and the more diseases you give [a person] the more chances that he will not survive. CX 1 at p.84.

Discussion of the Medical Opinion Evidence

The Board's instruction on remand is clear and narrow in scope. Specifically, I am to consider the opinions of Drs. Rizkalla and Perper and explain whether each physician has provided a reasoned opinion. In order to be credited, a medical report must be both reasoned and documented. *Trumbo v. Reading Anthracite Co.*, 17 B.L.R. 1-85 (1993). A documented opinion is one that sets forth the clinical findings, observations, facts, and other data upon which the

physician based the diagnosis. *Fields v. Island Creek Coal Co.*, 10 B.L.R. 1-19 (1987). A reasoned opinion is one in which the administrative law judge finds the underlying documentation and data adequate to support the physician's conclusions. *Fields, supra*. Indeed, whether a medical report is sufficiently documented and reasoned is for the judge as the finder-of-fact to decide. *Clark v. Karst-Robbins Coal Co.*, 12 B.L.R. 1-149 (1989)(*en banc*).

Dr. Perper opined that the immediate cause of death was acute bronchopneumonia that complicated the miner's chronic lung disease, which was due in significant part to his exposure to coal mine dust and resulting pneumoconiosis. He explained that the cause of the acute bronchopneumonia was the propensity of the miner to develop infectious disease because of his chronic lung disease that was primarily due to coal workers' pneumoconiosis and coal dust exposure, and that the miner's coal workers' pneumoconiosis was a substantial factor in causing, substantially contributing to, or hastening the miner's death.

Dr. Perper based his opinion on the miner's extensive medical histories and clinical information, the reports of other reviewing physicians including the autopsy prosector, and the autopsy slides. I find that his opinion is well documented. With respect to his reasoning, Dr. Perper meticulously explained, in both his medical opinion report and in his deposition testimony, how he arrived at his diagnoses and medical opinion. During his deposition testimony, he explained in logical and comprehensive detail the processes he found in the lung tissue and how they relate to the miner's overall clinical picture and his ultimate demise. He explained that while there is no typical symptom for a diagnosis of coal workers' pneumoconiosis, one must add to the symptoms the other elements, which include laboratory findings and especially pathological findings. To this end, Dr. Perper discussed various elements of the miner's clinical picture in relation to the pathology found on autopsy. In addition, he provided numerous references to medical literature in order to illustrate that the processes he identifies in his report are not only a result of his experience but also are recognized in the medical literature.

Dr. Perper also succinctly explained how both the miner's smoking history and his history of coal dust exposure both contributed to his centrilobular emphysema. When asked to separate the effects of the two, he admitted that it is not possible but explained that once a person stops smoking, there is no further deterioration of respiratory function except for age related deterioration and very slow progression, and he pointed out that the miner experienced a much more significant progression of respiratory symptoms after he stopped smoking. Although Dr. Perper testified that he considered between ten and thirty years of smoking history, even though some parts of the record indicate that the miner smoked forty or more years, he explained that thirty years was significant enough that he did not need to consider more time than that. In addition, Dr. Perper meticulously explained the process of aspiration pneumonia and his basis for discrediting it as the cause the of miner's pneumonia.

After reviewing Dr. Perper's medical opinion report and his deposition testimony, I find that his opinion is extremely well reasoned. Dr. Perper is very clear and definite in his opinion and when presented with the alternate theories and conflicting opinions of the opposing physicians, is very persuasive, thorough, and thoughtful in explaining why the evidence does not support their opinions. Where the evidence is not as clear-cut, Dr. Perper provides most credible

and logical explanations, and I find that his opinion is well supported by the underlying documentation and data. Consequently, I find that Dr. Perper more than thoroughly explained how the underlying documentation and data, both favorable and unfavorable, supports his diagnosis and that his opinion is extremely well reasoned and entitled to great weight.

Dr. Rizkalla opined that the miner had simple coal workers' pneumoconiosis with centrilobular emphysema and the immediate cause of death was terminal bronchopneumonia that complicated the coal workers' pneumoconiosis. He opined that coal workers' pneumoconiosis is a substantial contributing factor in the miner's death. As Dr. Rizkalla based his opinion on the miner's medical, social, and occupational histories, the reports of other physicians including the prosector, and the autopsy slides which he found to be of acceptable quality, I find that his opinion is well documented.

Like Dr. Perper, Dr. Rizkalla provided a detailed outline of the disease processes he identified in the miner's autopsy slides and related these processes to the miner's physical findings during his lifetime. Dr. Rizkalla also rejected the notion of aspiration pneumonia and provided a detailed explanation of both the process of aspiration pneumonia and why it should be rejected as a cause of the miner's death. He thoroughly explained that even if the particle seen in the photomicrographs was food, it would not be enough to change the miner's bronchopneumonia diagnosis to aspiration pneumonia, and he opined that the single foreign body material was so small that it could be pollen from the air.

Dr. Rizkalla ultimately opined that the miner developed terminal bronchopneumonia that complicated his emphysematous lungs, with the emphysema being part of the coal workers' pneumoconiosis. He explained that he could not exclude either the miner's cigarette smoking or his coal dust exposure from consideration as the cause of the emphysema. Dr. Rizkalla, however, relied on a forty-year occupational history and a twenty-year cigarette smoking history. The miner was credited with twenty-eight years of coal mine employment. ALJ Decision and Order at 9. Because Dr. Rizkalla relied on an inaccurate coal mine employment history, I find that it is entitled to less weight than it was originally given by ALJ Lesnick. *Worhach v. Director, OWCP*, 17 B.L.R. 1-105 (1993)(*per curiam*). Moreover, as Dr. Rizkalla opined that the emphysema, which was a part of the coal workers' pneumoconiosis, complicated the terminal bronchopneumonia, it would be helpful for me, as the trier-of-fact, to know whether the reduced occupational history might have changed his medical opinion as to the miner's demise. Accordingly, I find that Dr. Rizkalla's opinion is entitled to less weight.

Weighing the medical opinion evidence, I find that Dr. Perper's opinion is extremely well-reasoned, knowledgeable, and persuasive. The Board upheld ALJ Lesnick's findings that the contrary medical opinions were entitled to less weight. As I found to Dr. Perper's opinion to be particularly helpful and illustrative of the medical evidence and as it is the best-reasoned opinion in the record, I find that the claimant has established that the miner's death was due to pneumoconiosis pursuant to 20 C.F.R. § 718.205(c).

ORDER

The claim of RUTH L. BOWERSOX for benefits under the Act is AWARDED.

A

MICHAEL P. LESNIAK
Administrative Law Judge

NOTICE OF APPEAL RIGHTS: If you are dissatisfied with this Decision and Order you may file an appeal with the Benefits Review Board ("Board"). To be timely, your appeal must be filed with the Board within thirty (30) days from the date on which this Decision and Order is filed with the district director's office. *See* 20 C.F.R. §§ 725.478 and 725.479. The address of the Board is: ***Benefits Review Board, U.S. Department of Labor, P.O. Box 37601, Washington, D.C. 20013-7601.*** Your appeal is considered filed on the date it is received in the Office of the Clerk of the Board, unless the appeal is sent by mail and the Board determines that the U.S. Postal Service postmark, or other reliable evidence establishing the mailing date, may be used. *See* 20 C.F.R. § 802.207. Once an appeal is filed, all inquiries and correspondence should be directed to the Board.

After receipt of an appeal, the Board will issue a notice to all parties acknowledging receipt of the appeal and advising them as to any further action needed.

At the time you file an appeal with the Board, you must also send a copy of the appeal letter to Allen Feldman, Associate Solicitor for Black Lung and Longshore Legal Services, U.S. Department of Labor, 200 Constitution Avenue, N.W., Room N-2117, Washington, D.C. 20210. *See* 20 C.F.R. § 725.481.

If an appeal is not timely filed with the Board, this Decision and Order will become the final order of the Secretary of Labor pursuant to 20 C.F.R. § 725.479(a).